

(FILE 'HOME' ENTERED AT 18:56:41 ON 04 DEC 2002)

FILE 'MEDLINE, BIOSIS, CANCERLIT, LIFESCI, BIOTECHDS, CAPLUS' ENTERED AT
18:57:11 ON 04 DEC 2002

L1 32607 S LIF OR ((LEUKEMIA OR LEUKAEMIA) (W)INHIBIT? (W)FACTOR#)
L2 25279 S (DIFFERENTI?) (5A) (LEUKEMI# OR LEUKAEMI# OR ERYTHROLEUKEMI#
OR
L3 941 S L1 AND L2
L4 111 S L3 AND GLYCOPROTEIN
L5 48 DUP REM L4 (63 DUPLICATES REMOVED)
L6 340 S L1 AND AGGREGAT?
L7 79 S L1 (5A) AGGREGAT?
L8 295 S L6 AND PY<1999
L9 5824 S D(A) FACTOR
L10 85 S L9 AND AGGREGAT?
L11 1 S L6 AND (AMID? OR DEAMID?)
L12 0 S L10 AND (AMID? OR DEAMID?)
L13 117 S L1 (5A) (STABIL? OR (HALF(W)LIFE))
L14 17 S L9 (5A) (STABIL? OR (HALF(W)LIFE))
L15 134 S L13 OR L14
L16 105 S L15 AND PY<1999
L17 93 DUP REM L16 (12 DUPLICATES REMOVED)
L18 3 S L17 AND (LEUKEMI# OR ERYTHROLEUKEMI# OR LEUKAEMI# OR ERYTHRO

L18 ANSWER 2 OF 3 MEDLINE
 ACCESSION NUMBER: 81209972 MEDLINE
 DOCUMENT NUMBER: 81209972 PubMed ID: 6972252
 TITLE: Effect of tunicamycin on production by mouse fibroblast
 L929 cells of the factor-stimulating differentiation of
 mouse myeloid **leukemic** cells and the
 colony-stimulating factor.
 AUTHOR: Yamamoto Y; Tomida M; Hozumi M; Ayusawa D; Seno T; Tamura
 G
 SOURCE: CANCER RESEARCH, (1981 Jun) 41 (6) 2534-9.
 Journal code: 2984705R. ISSN: 0008-5472.
 PUB. COUNTRY: United States
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 198108
 ENTRY DATE: Entered STN: 19900316
 Last Updated on STN: 19980206
 Entered Medline: 19810827

AB Mouse myeloid **leukemic** M1 cells can be induced to differentiate
 into macrophages and granulocytes in vitro by a factor(s) stimulating
 differentiation of the cells (D-factor), which is suggested to be a
 glycoprotein. On the other hand, growth and differentiation of normal
 precursor cells of macrophages and granulocytes can be stimulated by a
 glycoprotein termed colony-stimulating factor (CSF). Mouse fibroblast
 L929
 cells were found to produce both the D-factor and CSF. The properties of
 the D-factor and CSF and the roles of carbohydrates in the molecules of
 these factors were examined using tunicamycin, a specific inhibitor of
 asparaginase-linked glycosylation. Although both the D-factor and CSF
 were
 produced by L-cells in usual medium containing fetal calf serum,
 production of D-factor, but not CSF, was reduced by omission of serum
 from
 the medium. The activity of the D-factor was slightly decreased by
 treating the L-cells with tunicamycin (0.5 microgram/ml) in the presence
 of 2% fetal calf serum, without any decrease in CSF activity. Conditioned
 medium of L-cells incubated with or without tunicamycin was fractionated
 by gel filtration on a Sephadex G-200 column. Normal D-factor appeared as
 a single peak with an apparent molecular weight of 67,000. D-factor
 produced in the presence of tunicamycin had an apparent molecular weight
 of 25,000. On the other hand, most of the CSF was eluted in the void
 volume, even when it was produced in the presence of tunicamycin. The
 D-factor produced in the presence of tunicamycin was more sensitive than
 normal D-factor was to trypsin or heat treatment at 70 degrees. The CSF
 produced in the presence of tunicamycin was resistant to these
 treatments.
 These results indicate that the D-factor is distinct from CSF.
 Furthermore, the results suggest that the D-factor produced by L-cells is
 also a glycoprotein and that, although carbohydrate is not essential for
 production or activity of the **D-factor**, it contributes
 to **stabilizing** the protein portion of **D-factor**

L5 ANSWER 48 OF 48

MEDLINE

DUPLICATE 24

ACCESSION NUMBER: 89048312 MEDLINE

DOCUMENT NUMBER: 89048312 PubMed ID: 3142300

TITLE: Purification of a murine **leukemia inhibitory factor** from Krebs ascites cells.

AUTHOR: Hilton D J; Nicola N A; Metcalf D

CORPORATE SOURCE: Walter and Eliza Hall Institute of Medical Research, Royal Melbourne Hospital Victoria, Australia.

SOURCE: ANALYTICAL BIOCHEMISTRY, (1988 Sep) 173 (2) 359-67.
Journal code: 0370535. ISSN: 0003-2697.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 198812

ENTRY DATE: Entered STN: 19900308

Last Updated on STN: 19970203

Entered Medline: 19881214

AB A factor capable of inducing terminal **differentiation** in the murine myeloid **leukemia** cell line M1 has been purified to apparent homogeneity from the medium conditioned by Krebs II ascites tumor

cells. The factor, termed **leukemia inhibitory factor (LIF)** is a single chain **glycoprotein** of apparent Mr 58,000 which induces differentiation and inhibits proliferation of the M1 cell line but not the WEHI-3B D+ murine myeloid leukemic cell line and has no detectable proliferative activity on normal myeloid progenitor cells. It was purified using four successive high-efficiency purification steps--anion-exchange chromatography on DEAE-Sepharose; cation-exchange chromatography on CM-Sepharose; affinity chromatography on lentil lectin-Sepharose; and reverse-phase high-performance liquid chromatography on a phenyl-silica matrix--to a specific biological activity of approximately 1.25×10^8 units/mg with an overall purification of 12,000-fold and a yield of 73% for the activity

failing to bind to DEAE-Sepharose. Sufficient quantities of the factor (12

micrograms, 200 pmol) have been purified to allow structural and functional analysis of the molecule and comparison with other known differentiation inducers.

L5 ANSWER 46 OF 48 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1988:376457 BIOSIS
DOCUMENT NUMBER: BA86:60367
TITLE: RESOLUTION AND PURIFICATION OF THREE DISTINCT FACTORS
PRODUCED BY KREBS ASCITES CELLS WHICH HAVE

DIFFERENTIATION-INDUCING ACTIVITY ON MURINE MYELOID
LEUKEMIC CELL LINES.

AUTHOR(S): HILTON D J; NICOLA N A; GOUGH N M; METCALF D
CORPORATE SOURCE: WALTER AND ELIZA HALL INST. MED. RES., P.O. ROYAL
MELBOURNE

HOSP. 3050, VICTORIA, AUST.

SOURCE: J BIOL CHEM, (1988) 263 (19), 9238-9243.
CODEN: JBCHA3. ISSN: 0021-9258.

FILE SEGMENT: BA; OLD

LANGUAGE: English

AB The use of different myeloid leukemic cell lines (WEHI-3B D+ and M1) and different sources of factors has led to discrepancies concerning the identity of factors capable of inducing **differentiation** in **leukemic** cells. We have biochemically fractionated medium conditioned by one such source (Krebs II ascites cells) and assayed fractions for their bone marrow colony-stimulating activity as well as their differentiation-inducing activity for WEHI-3B D+ and M1 cells. This resulted in the resolution of four distinct molecular species with differentiation-inducing activity. One activity was purified to homogeneity and shown by a variety of biochemical, biological, and receptor-binding criteria to be authentic granulocyte colony-stimulating factor (G-CSF). A second activity was identified as granulocyte-macrophage colony-stimulating factor (GM-CSF). Two other activities termed **LIF-A** and **LIF-B (leukemia inhibitory factor)** were shown to probably be different glycosylation variants of the same protein and one of these (**LIF-A**) was purified 12,000-fold to homogeneity. G-CSF induced differentiation in both WEHI-3B D+ and at higher concentrations M1 cells while GM-CSF weakly induced differentiation in WEHI-3B D+ cells. **LIF-A** had no colony-stimulating activity and induced differentiation in and inhibited the proliferation of only M1 cells. Each factor bound to a unique cell surface receptor with no evidence of direct cross-reactivity.

L5 ANSWER 44 OF 48 MEDLINE DUPLICATE 21
 ACCESSION NUMBER: 90200634 MEDLINE
 DOCUMENT NUMBER: 90200634 PubMed ID: 2180647
 TITLE: Protein factors that regulate the growth and
 differentiation of mouse myeloid **leukaemia**
 cells.
 AUTHOR: Hozumi M; Tomida M; Yamamoto-Yamaguchi Y; Kasukabe T;
 Okabe-Kado J; Honma Y; Hayashi M
 CORPORATE SOURCE: Department of Chemotherapy, Saitama Cancer Center Research
 Institute, Japan.
 SOURCE: CIBA FOUNDATION SYMPOSIUM, (1990) 148 25-33; discussion
 33-42. Ref: 28
 Journal code: 0356636. ISSN: 0300-5208.
 PUB. COUNTRY: Netherlands
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 General Review; (REVIEW)
 (REVIEW, TUTORIAL)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199005
 ENTRY DATE: Entered STN: 19900601
 Last Updated on STN: 19900601
 Entered Medline: 19900508

AB We have purified and characterized several protein factors that regulate
 the growth and **differentiation** of mouse myeloid
 leukaemia M1 cells. The **differentiation** factor
 (D-factor) from conditioned medium (CM) of Ehrlich ascites tumour cells

is

a **glycoprotein** of Mr 40,000-50,000. Its amino acid sequence was
 found to be almost identical to that of **leukaemia**
inhibitory factor (LIF) from Krebs II ascites
 cells. The differentiation inhibitory factor (I-factor) from the CM of
 variant M1 cell clones which were resistant to several differentiation
 inducers is a basic protein of apparent Mr 68,000. The growth inhibitory
 factor (GI-factor) that specifically inhibits the partially
differentiated and still growing monocytic **leukaemia** M1
 cells was isolated from the CM of a clone of M1 cells resistant to the
 differentiation inducers. This GI-factor is a basic protein with an Mr of
 25,000. Regulation by these protein factors together with other known
 cytokines of growth and differentiation of M1 cells is reported.

L5 ANSWER 42 OF 48 MEDLINE

ACCESSION NUMBER: 90253815 MEDLINE

DOCUMENT NUMBER: 90253815 PubMed ID: 2111155

TITLE: Localization of the murine **leukemia inhibitory factor** gene near the centromere on chromosome 11.

AUTHOR: Kola I; Davey A; Gough N M

CORPORATE SOURCE: Centre for Early Human Development, Monash Medical Center, Clayton, Victoria, Australia.

CONTRACT NUMBER: CA-22556 (NCI)

SOURCE: GROWTH FACTORS, (1990) 2 (2-3) 235-40.
Journal code: 9000468. ISSN: 0897-7194.

PUB. COUNTRY: Switzerland

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199006

ENTRY DATE: Entered STN: 19900720

Last Updated on STN: 19900720

Entered Medline: 19900625

AB **Leukemia inhibitory factor (LIF)**

is a **glycoprotein** with divergent activities: It induces the **differentiation** of certain myeloid **leukemic** cells, inhibits the **differentiation** of embryonic stem cells, and promotes bone remodelling in vivo and in vitro. The murine **LIF** gene has been assigned to the proximal region of chromosome 11 at sub-bands A1-A2, by analysis of a panel of mouse x Chinese hamster somatic

cell hybrids and by in situ hybridization. Interestingly, the proximal portion of chromosome 11 has been shown, by virtue of its parental origin effects, to contain gene(s) involved in fetal growth. It is also interesting that there is a preponderance of chromosome 11 abnormalities in embryonal carcinoma cells. The localization of the murine **LIF** gene confirms the homology of a portion of murine chromosome 11 with

human

chromosome 22q, the site of the human **LIF** gene.

L5 ANSWER 41 OF 48 LIFESCI COPYRIGHT 2002 CSA

ACCESSION NUMBER: 93:100886 LIFESCI

TITLE: Developmentally programmed induction of differentiation inhibiting activity and the control of stem cell populations.

AUTHOR: Rathjen, P.D.; Nichols, J.; Toth, S.; Edwards, D.R.; Heath,

J.K.; Smith, A.G.

CORPORATE SOURCE: CRC Growth Factors Group, Dep. Biochem., Univ. Oxford, Oxford OX1 3QU, UK

SOURCE: GENES DEV., (1990) vol. 4, no. 126, pp. 2308-2318.

DOCUMENT TYPE: Journal

FILE SEGMENT: F

LANGUAGE: English

SUMMARY LANGUAGE: English

AB **Differentiation** inhibiting activity/**leukemia**

inhibitory factor (DIA/**LIF**) is a **glycoprotein** that controls differentiation of pluripotential stem cells. Alternative transcription generates both diffusible and matrix-associated forms of DIA/**LIF**. Transcriptional analysis using a sensitive ribonuclease protection assay revealed that the two messages are expressed independently, consistent with the proposition that the two forms of DIA/**LIF** have distinct biological roles. DIA/**LIF** expression was found to be activated early during differentiation of embryonic stem (ES) cells, providing a mechanism for feedback regulation of stem cell renewal. Expression of DIA/**LIF** by mesenchymal cells was shown to be controlled in a paracrine manner by polypeptide regulatory factors.

L5 ANSWER 40 OF 48 MEDLINE DUPLICATE 19
ACCESSION NUMBER: 91122633 MEDLINE
DOCUMENT NUMBER: 91122633 PubMed ID: 1703981
TITLE: Developmentally programmed induction of differentiation
inhibiting activity and the control of stem cell
populations.
AUTHOR: Rathjen P D; Nichols J; Toth S; Edwards D R; Heath J K;
Smith A G
CORPORATE SOURCE: Department of Biochemistry, University of Oxford, UK.
SOURCE: GENES AND DEVELOPMENT, (1990 Dec) 4 (12B) 2308-18.
Journal code: 8711660. ISSN: 0890-9369.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199103
ENTRY DATE: Entered STN: 19910405
Last Updated on STN: 19960129
Entered Medline: 19910314

AB **Differentiation** inhibiting activity/**leukemia**
inhibitory factor (DIA/**LIF**) is a
glycoprotein that controls differentiation of pluripotential stem
cells. Alternative transcription generates both diffusible and
matrix-associated forms of DIA/**LIF**. Transcriptional analysis
using a sensitive ribonuclease protection assay revealed that the two
messages are expressed independently, consistent with the proposition
that
the two forms of DIA/**LIF** have distinct biological roles. DIA/
LIF expression was found to be activated early during
differentiation of embryonic stem (ES) cells, providing a mechanism for
feedback regulation of stem cell renewal. Expression of DIA/**LIF**
by mesenchymal cells was shown to be controlled in a paracrine manner by
polypeptide regulatory factors. Specific expression of the two forms of
DIA/**LIF** was also demonstrated in the egg cylinder-stage mouse
embryo. The combination of cell type-specific and signal-specific
regulation enables very precise control over DIA/**LIF** expression
and may represent an important component of the regulatory networks that
govern stem cell proliferation and differentiation during mammalian
development.

L5 ANSWER 35 OF 48 MEDLINE DUPLICATE 15
ACCESSION NUMBER: 92020908 MEDLINE
DOCUMENT NUMBER: 92020908 PubMed ID: 1717982
TITLE: Oncostatin M is a member of a cytokine family that
includes

leukemia-inhibitory factor,
granulocyte colony-stimulating factor, and interleukin 6.
AUTHOR: Rose T M; Bruce A G
CORPORATE SOURCE: Fred Hutchinson Cancer Research Center, Seattle, WA
98104.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE
UNITED STATES OF AMERICA, (1991 Oct 1) 88 (19) 8641-5.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199110
ENTRY DATE: Entered STN: 19920124
Last Updated on STN: 19960129
Entered Medline: 19911031

AB Oncostatin M (OSM), a **glycoprotein** of Mr approximately 28,000
produced by activated monocyte and T-lymphocyte cell lines, was
previously
identified by its ability to inhibit the growth of cells from melanoma
and
other solid tumors. We have detected significant similarities in the
primary amino acid sequences and predicted secondary structures of OSM,
leukemia-inhibitory factor (LIF),
granulocyte colony-stimulating factor (G-CSF), and interleukin 6 (IL-6).
Analysis of the genes encoding these proteins revealed a shared exon
organization, suggesting evolutionary descent from a common ancestral
gene. Using a panel of DNAs from somatic cell hybrids, we have shown that
OSM, like **LIF**, is located on human chromosome 22. We have also
demonstrated that OSM has the ability to inhibit the proliferation of
murine M1 myeloid **leukemic** cells and can induce their
differentiation into macrophage-like cells, a function shared by
LIF, G-CSF, and IL-6. We propose that OSM, **LIF**, G-CSF,
and IL-6 are structurally related members of a cytokine family that have
in common the ability to modulate differentiation of a variety of cell
types.

L5 ANSWER 29 OF 48 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1994:75172 CAPLUS

DOCUMENT NUMBER: 120:75172

TITLE: Soluble interleukin-6 receptor triggers osteoclast formation by interleukin 6

AUTHOR(S): Tamura, Tatsuya; Udagawa, Nobuyuki; Takahashi, Naoyuki; Miyaura, Chisato; Tanaka, Sakae; Yamada, Yoshiki; Koishihara, Yasuo; Ohsugi, Yoshiyuki;

Kumaki,

Kenji; et al.

CORPORATE SOURCE: Sch. Dent., Showa Univ., Tokyo, 142, Japan

SOURCE: Proceedings of the National Academy of Sciences of the

United States of America (1993), 90(24), 11924-8

CODEN: PNASA6; ISSN: 0027-8424

DOCUMENT TYPE: Journal

LANGUAGE: English

AB It has been reported that sol. interleukin (IL)-6 receptor (sIL-6R) is detected in the serum of healthy individuals and its level is increased in

patients with multiple myeloma and human immunodeficiency virus infection.

Although several reports have suggested that sIL-6R potentiates IL-6 action, its physiol. role remains unclear. In this study, the authors examd. the role of sIL-6R on osteoclast formation by IL-6, using a coculture of mouse osteoblasts and bone marrow cells. Neither recombinant

mouse IL-6 (mIL-6) nor mouse sIL-6R (smIL-6R) induced osteoclast-like multinucleated cell (MNC) formation when they were added sep. In contrast, simultaneous treatment with mIL-6 and smIL-6R strikingly induced

MNC formation. These MNCs satisfied major criteria of authentic osteoclasts, such as tartrate-resistant acid phosphatase (TRAP) activity, calcitonin receptors, and pit formation on dentin slices. The MNC formation induced by mIL-6 and smIL-6R was dose-dependently inhibited by adding monoclonal anti-mouse IL-6R antibody (MR16-1). It is likely that osteoblasts and osteoclast progenitors are capable of transducing a signal

from a complex of IL-6 and sIL-6R through gp130, even though they may have

no or a very small no. of IL-6Rs. Factors such as IL-11, oncostatin M, and **leukemia inhibitory factor**, which are known to exert their functions through gp130 (the signal-transducing chain

of IL-6R), also induced MNC formation in the authors' coculture system. These results suggest that increased circulating or locally produced sIL-6R induces osteoclast formation in the presence of IL-6 mediated by a mechanism involving gp130. This may play an important physiol. or pathol.

role in conditions assocd. with increased osteoclastic bone resorption.

L5 ANSWER 21 OF 48 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1997:292859 CAPLUS

DOCUMENT NUMBER: 126:329104

TITLE: Regulation of oligodendrocyte cell survival and
differentiation by ciliary neurotrophic
factor, **leukemia inhibitory**

AUTHOR(S): Vos, J. P.; Gard, A. L.; Pfeiffer, S. E.

CORPORATE SOURCE: Department of Microbiology, University of Connecticut
Medical School, Farmington, CT, USA

SOURCE: Perspectives on Developmental Neurobiology (1996),
4(1), 39-52, 1 Plate

CODEN: PDENED; ISSN: 1064-0517

PUBLISHER: Gordon & Breach

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review with 133 refs. The regulation and maintenance of developmental
lineages by trophic factors, both cell-mediated and sol., is a key aspect
of cellular differentiation in the nervous system. In this review we
focus on oligodendrocytes and their progenitors and how differentiation
and survival are regulated by four neuropoietic cytokines: ciliary
neurotrophic factor, **leukemia inhibitory**

factor, oncostatin M, and interleukin-6 (IL-6). We discuss how
these cytokines act as "broad spectrum" factors. I.e., how, even within

a
specific cell lineage, a given cytokine may have different effects on the
target cells at various stages of differentiation.

L5 ANSWER 22 OF 48 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1995:556801 CAPLUS

DOCUMENT NUMBER: 122:288671

TITLE: Cardiotrophin-1. Biological activities and binding to the **leukemia inhibitory factor** receptor/gp130 signaling complex

AUTHOR(S): Pennica, Diane; Shaw, Kenneth J.; Swanson, Todd A.; Moore, Mark W.; Shelton, David L.; Zioncheck, Kimberly

A.; Rosenthal, Arnon; Taga, Tetsuya; Paoni, Nicholas F.; Wood, William I.

CORPORATE SOURCE: Dep. Molecular Biol., Cell Genetics, Neurobiol., Immunology, South San Francisco, CA, 94080, USA

SOURCE: Journal of Biological Chemistry (1995), 270(18), 10915-22

CODEN: JBCHA3; ISSN: 0021-9258

PUBLISHER: American Society for Biochemistry and Molecular Biology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Cardiotrophin-1 (CT-1) is a newly isolated cytokine that was identified based on its ability to induce cardiac myocyte hypertrophy. It is a member of the family of cytokines that includes interleukins-6 and -11, **leukemia inhibitor factor (LIF)**, ciliary neurotrophic factor, and oncostatin M. These cytokines induce a pleiotropic set of growth and differentiation activities via receptors that use a common signaling subunit, gp130. In this work we det. the activity of CT-1 in six in vitro biol. assays and examine the compn. of its cell surface receptor. We find that CT-1 is inactive in stimulating the growth of the hybridoma cell line, B9 and inhibits the growth of the mouse myeloid leukemia cell line, M1. CT-1 induces a phenotypic switch

in rat sympathetic neurons and promotes the survival of rat dopaminergic and chick ciliary neurons. CT-1 also inhibits the differentiation of mouse embryonic stem cells. CT-1 and **LIF** cross-compete for binding to M1 cells, Kd [CT-1] .apprx. 0.7 nM, and this binding is inhibited by an anti-gp130 monoclonal antibody. Both ligands can be specifically cross-linked to a protein on M1 cells with the mobility of the **LIF** receptor (.apprx.200 kDa). In addn., CT-1 binds directly to a purified, sol. form of the **LIF** receptor in soln. (Kd .apprx. 2 nM). These data show that CT-1 has a wide range of hematopoietic, neuronal, and developmental activities and that it can act via the **LIF** receptor and the gp130 signaling subunit.

L5 ANSWER 7 OF 48 MEDLINE DUPLICATE 4
 ACCESSION NUMBER: 2000078517 MEDLINE
 DOCUMENT NUMBER: 20078517 PubMed ID: 10613356
 TITLE: Induction of gp130 and **LIF** by
 differentiation inducers in human myeloid
 leukemia K562 cells.
 AUTHOR: Xie P; Chan F S; Ip N Y; Leung M F
 CORPORATE SOURCE: Department of Biology, Biotechnology Research Institute,
 The Hong Kong University of Science and Technology,
 People's Republic of China.
 SOURCE: LEUKEMIA RESEARCH, (1999 Dec) 23 (12) 1113-9.
 Journal code: 7706787. ISSN: 0145-2126.
 PUB. COUNTRY: ENGLAND: United Kingdom
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 200001
 ENTRY DATE: Entered STN: 20000124
 Last Updated on STN: 20000124
 Entered Medline: 20000110
 AB It has been previously shown that phorbol 12-myristate 13-acetate (PMA),
 a potent differentiation inducer, induced the expression of both
 interleukin-6 (IL-6) and IL-6 receptor alpha component (IL-6Ralpha) in
 K562 leukemia cells. In the present study, we examined the ability of
 several differentiation inducers to regulate the expression of the
 signal-transducing receptor component for IL-6, gp130, and cytokine
 leukemia inhibitory factor (LIF) in
 K562 cells. We found that the expression of gp130 was dramatically
 induced
 at both the mRNA and protein levels by the two megakaryocytic inducers
 sodium butyrate (NaBut) and PMA. In contrast, the mRNA expression of
 LIF was induced by the two erythroid inducers 1-beta-D-
 arabinofuranosyl cytosine (Ara-C) and hemin. Furthermore, activation of
 the PMA-induced gp130 receptor by exogenous IL-6 potentiated the
 differentiating effects of PMA. Our findings suggest that IL-6/gp130
 signaling may be involved in the regulation of the megakaryocytic
 differentiation of K562 cells.